Expert Witness Disclosure of William G. Hughson, M.D., D. Phil.

I submit this report on behalf of W.R. Grace & Co., et al. ("Debtors").

I. Background and Qualifications

I am the Director of the University of California San Diego ("UCSD") Center for Occupational and Environmental Medicine and Clinical Professor of Medicine at UCSD Medical Center, 200 West Arbor Drive, San Diego, California 92103-8800. I am also an Adjunct Associate Professor at the San Diego State University Graduate School of Public Health.

I am a medical doctor and am board certified in internal medicine, pulmonary medicine, and occupational medicine. I received a doctorate degree in epidemiology from Oxford University, where I studied as a Rhodes Scholar under Sir Richard Doll, one of the foremost epidemiologists in the world.

I am a member of numerous professional organizations, including the American College of Chest Physicians, the American College of Physicians, the American Thoracic Society, the American College of Occupational & Environmental Medicine, the Western Occupational and Environmental Medical Association, and the Royal College of Physicians and Surgeons of Canada. I have served as a member of numerous committees of those organizations concerning occupational and environmental medicine issues.

I have had extensive experience in the evaluation and treatment of patients who have been exposed to asbestos. For example, I administered a program to screen workers at the National Steel and Shipbuilding Company (NASSCO) in San Diego for asbestos-related diseases, and performed approximately 1,000 evaluations of NASSCO workers. In addition, since 1985, I have served as the UCSD Medical Director for Employee Health Services with responsibility for monitoring the health of workers exposed to asbestos containing materials and

advising with respect to removal and renovation projects. I have also been involved in screening and evaluation programs for various other workers with potential asbestos exposure.

I have extensively reviewed the medical literature concerning asbestos-related disease. I keep abreast of the current scientific and medical literature on the subject by reading numerous academic journals, undertaking continuing medical education, and accessing other available materials through the National Library of Medicine. All of this reading over the course of my professional career, together with my actual clinical experience and supervisory responsibilities relating to asbestos, provide the basis for my expert opinions as expressed in this report.

A more complete statement of my background, professional qualifications and experience is set forth in the attachments to this statement. A copy of my current curriculum vitae ("Attachment A") lists my publications. My prior deposition/trial testimony for the last four years is also attached ("Attachment B"). Each of the opinions expressed herein is held within a reasonable degree of scientific and medical certainty.

II. Types of Asbestos Fibers

There are two major types of asbestos fibers – serpentine and amphibole. The former contains curvilinear asbestos fibers. A sub-category of serpentine asbestos fibers is chrysotile asbestos, commonly referred to as "white asbestos." Most chrysotile asbestos came from the Quebec province in Canada. Approximately 95 percent of the asbestos used in the United States was chrysotile.

The second major category of asbestos fibers is amphibole. Amphibole fibers are straight. One sub-category of amphibole asbestos is amosite, often referred to as "brown asbestos." Amosite usually comes from South Africa, and has been mostly used in shipyard applications. Another sub-category of amphibole asbestos is crocidolite, often called "blue

asbestos." Crocidolite typically comes from South Africa and Australia. Tremolite is also a type of amphibole, and although it has not been used in North America for commercial purposes, tremolite has been found as a natural contaminant of chrysotile asbestos and vermiculite.

III. The Body's Defense Mechanisms Against Airborne Particulate Dust Matter

The human body has several defense mechanisms that assist in trapping and expelling inhaled particulate dust matter. The body's first defense mechanism against airborne particulate dust matter, including asbestos fibers, is the nose. The nose contains turbinates and a mucus membrane. Turbinates spin the air as it is inhaled, and also heat and humidify the inhaled air. Particulate dust matter adheres to the mucus membrane in the nose and is expelled via a cough, sneeze or by being swallowed and cleared through the gastrointestinal system. By the time inhaled air reaches the lungs, the majority of particulate dust matter has been stopped by these mechanisms.

Upon inhalation, air enters the trachea and then the left and right main stem bronchi. The bronchi continue to branch into smaller and smaller bronchioles which terminate in the alveoli. The bronchi and central bronchioles are lined with ciliated epithelium and mucus producing cells.

The particulate dust matter which enters the lungs, if it has not been trapped by the nasal mucus membrane, may be trapped by the mucus producing cells lining the bronchi and bronchioles. These ciliated cells sweep the mucus with any trapped particulate dust matter upwards, causing a cough or sneeze. Particulate dust matter is then expelled from the respiratory system. This process is referred to as the mucociliary escalator.

Macrophages are another defense mechanism against airborne particulate dust matter. Part of the immune system, macrophages are produced in the bone marrow. Once mature,

macrophages are transported from the bone marrow into the lungs by the bloodstream. Once in the lung, macrophages migrate through the blood vessel wall and the interstitial layer and enter the air spaces of the lung. Macrophages recognize inhaled dust and particulate matter as foreign substances and ingest the dust and particulate matter. This process is called phagocytosis.

Finally, if particulate dust matter escapes the body's other defense mechanisms, some are then cleared away by the lymphatic system, which may or may not include an interaction with macrophages.

Despite the body's very efficient defense mechanisms all particulate dust matter will not be cleared from the respiratory system. Some dust fibers can remain in the lung indefinitely and could move out of the airways into the parenchyma through a process of migration via the epithelial layer. Some of the inhaled fibers are coated with iron-containing protein. These coated fibers are visible on microscopic examination and are referred to as asbestos bodies or ferruginous bodies.

Animal studies demonstrate that approximately 75% of inhaled asbestos fibers are cleared and then found in the feces of the animal within 24 to 48 hours. Another percentage of fibers are deposited more deeply in the lungs, but are subsequently cleared. A residual number of fibers, probably 5-10% of inhaled asbestos fibers, are not cleared by natural defense mechanisms within the first 30 days following inhalation.

IV. Diseases Epidemiologically Established to be Related to Asbestos Exposure

A. Nonmalignant Diseases Associated with Exposure to Asbestos

Inhalation of asbestos fibers ("asbestos exposure") is associated with both nonmalignant and malignant diseases. The primary nonmalignant disease associated with asbestos is asbestosis. Asbestosis is manifest by lung scarring due to inhalation of asbestos dust that is not cleared by the

lung's defense mechanisms. Individuals, however, must have a high level of asbestos exposure over a significant period of time before they develop asbestosis.

Asbestos can also cause scarring in the pleura. The pleura consists of a layer of mesothelial cells which cover the lung and the inside of the chest wall. Areas of pleural scarring are called pleural plaques. Pleural plaques usually do not cause symptoms or impairment unless they are very extensive.

Asbestos can also cause pleural effusions, which are collections of fluid that accumulate between the lung and the chest wall.

B. Malignant Diseases Associated with Exposure to Asbestos

The malignancies associated with asbestos exposure are lung cancer and mesothelioma. Lung cancer, like asbestosis, requires a high level of asbestos exposure over a significant period of time because one must first develop asbestosis before developing asbestos-induced lung cancer. Individuals with asbestosis who smoke increase their risk of developing asbestos-related lung cancer.

Mesothelioma is a tumor arising either in the lining of the chest, which is called the pleura, or the lining of the abdomen, which is called the peritoneum. Mesothelioma is predominantly associated with occupational exposure to asbestos. Asbestos exposure, however, is not the only cause of mesothelioma. Other causes include radiation, erionite, and chronic inflammation. Approximately 25% occur in people with no history of asbestos exposure, or any of the other recognized causes of mesothelioma. Given the other recognized causes of mesothelioma, and the significant percentage of mesothelioma cases that are idiopathic, the opinion that asbestos exposure is the only generally recognized cause of mesothelioma is erroneous.

While it is accepted that asbestos causes mesothelioma and lung cancer under certain conditions of exposure, there is dispute in the scientific community as to whether asbestos causes other cancers. There is some evidence that asbestos exposure increases the risk of laryngeal cancer, but that literature is confounded by the effects of smoking and alcohol. There is insufficient evidence to conclude that asbestos causes urinary or gastrointestinal cancers. (Goodman M, Morgan RW, Ray R, et al. Cancer in asbestos-exposed occupational cohorts: a meta analysis. Cancer Causes and Control 1999;10:453-465. Samet JM, Bristow LR, Clarkson TW, et al. Asbestos: Selected Cancers. Committee on Asbestos: Selected Health Effects. Institute of Medicine of the National Academies. The National Academies Press, 2006.)

V. The Relationship Between Dose, Fiber Type, and Fiber Size and the Development of Asbestos-Related Disease

To analyze the risk of asbestos exposure, it is important to consider dose, the type of asbestos, and the physical dimensions of the fibers. Dose is a function of both intensity and duration of exposure. Fiber type refers to whether the asbestos is an amphibole (e.g., amosite, crocidolite, or tremolite) or serpentine (e.g., chrysotile). Fiber size impacts the development of disease because only fibers of certain dimensions are respirable, and/or have been epidemiologically found to increase the risk of developing an asbestos-related disease.

A. Dose

There is general agreement in the medical and scientific literature that the risk of developing an asbestos-related disease is related to the amount of asbestos exposure, i.e. the dose. (Liddell FDK, McDonald AD, McDonald JC., The 1891-1920 birth cohort of Quebec chrysotile miners and millers; development from 1904 and mortality to 1992. Ann Occup Hyg

1997; 41:13-35). Furthermore, the severity of disease and the risk of its progression are also dose-related. (Jakobsson K, Stomber U, Albin M, et al., Radiological changes in asbestos cement workers. Occup Environ Med 1995; 52:20-27).

Dose, which is normally expressed in terms of fibers per cubic centimeter years ("f/cc-years"), takes into account both the intensity and the duration of the exposure to asbestos. Intensity, which is usually expressed in terms of fibers per cubic centimeter of air ("f/cc"),[1] refers to the concentration of airborne asbestos fibers in the breathing zone. Duration measures the length of exposure, that is, the period of time a person is exposed to a particular intensity. Duration is usually expressed in terms of working years (250 days x 8 hours = 2000 hours).

The epidemiological literature on asbestos normally considers or reflects cumulative life-time exposures to asbestos. Cumulative life-time exposure is a calculation of the time-weighted average of the exposure intensity times the number of years that a population (or a person) was exposed at that level. This result is expressed as f/cc-years.[2] For example, in an atmosphere where the time-weighted average intensity of asbestos is 1 f/cc, an individual would accumulate a 1 f/cc-year dose after one year of exposure at that level. As another example, an asbestos insulator, working in an environment with 10 f/cc exposure intensity for 20 years, would have a dose of 200 f/cc-years.

A time-weighted average reflects the fact that, in the workplace, exposure to asbestos normally is not constant for the work day, but typically varies throughout the day. The time-

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1 Fiber per cubic centimeter ("f/cc") and fiber per cubic centimeter years ("f/cc year") are the measurements for intensity and dose that are repeatedly referenced in the epidemiological literature on asbestos-related diseases. Earlier literature and studies, however, referenced millions of particles per cubic foot ("MPPCF"), another measure of intensity.

2 In deriving fibers/cc year it is assumed that an individual works eight hours a day and that the work year is 250 days.

weighted average accounts for peak exposures and low exposures, and permits both to be considered in determining an individual's cumulative exposure.

To calculate a time-weighted average, the different concentrations of exposure throughout a day must be determined. Once those concentrations of exposure are determined, the time in minutes for which an individual is exposed to each concentration is determined. The time (in minutes) for each level of exposure must equal the total number of minutes in an eight hour day, that is, 480 minutes. The time in minutes for each concentration level is multiplied by that concentration, and all sums are added together, resulting in a time-weighted average.

B. Fiber Type

There is evidence that amphibole fibers (on a per-unit-dose basis) create a greater risk for asbestosis and lung cancer than chrysotile fibers. Although amphibole fibers tend to be retained in the lung for a longer duration, the different amphibole fibers are not equal in potency. Crocidolite is the amphibole that creates the greatest asbestos-disease risk, followed by amosite. Tremolite has approximately the same risk as amosite. (Hodgson JT, Darnton A. The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. Ann Occup Hyg 2000;44:565-601.) Amphibole fibers are not cleared as easily by the body's defense mechanisms as chrysotile fibers. This is because chrysotile fibers tend to be dissolved or broken into smaller pieces, making it easier for the body's defense mechanisms to clear them.

C. Fiber Size

Fiber size is an important consideration in the determination of what effect, if any, asbestos exposure may have on an individual. Fiber size determines whether a fiber will be respirable or not. Dust particles and asbestos fibers with a mean aerodynamic diameter (MAD)

greater than 10 microns will not enter the lungs. Dust particles and asbestos fibers with a MAD of 1-3 microns may be inhaled and subsequently deposited into the lungs.

The human body's immune system evolved to handle natural infections such as viruses and bacteria that are a few microns in length. Bacteria can only be two or three microns in length. A macrophage, therefore, has the capability of ingesting bacteria. Asbestos fibers exist in varying lengths, and the body's defense systems are unable to destroy very long fibers (e.g. >10 microns). Animal data, as well as human data, suggests that long, thin and durable fibers are not as easily cleared by the body's defense mechanisms and that the presence of these types of fibers in the lungs may result in inflammation of the lung tissue.

The issue of fiber size has been reviewed by an advisory panel to the EPA, who concluded that the risk for fibers less than 5 microns in length was very low, and could be zero. (Report on the Peer Consultation Workshop to Discuss a Proposed Protocol to Assess Asbestos-Related Risk. Prepared for the U.S. Environmental Protection Agency, Office of Solid Waste and Emergency Response. Washington DC. EPA Contract No. 68-C-98-148. Work Assignment 2003-2005. Final Report May 30, 2003.)

VI. Principles of Epidemiology

Epidemiology is the study of the incidence, distribution, and control of disease in a specific population. The development of information concerning the risk of disease typically begins with the publication of case reports describing unusual medical conditions. These case reports attract the interest of scientists, triggering the performance of formal epidemiological studies, including "retrospective" and "prospective" epidemiological studies.

Case reports are generally reports describing the incidence of a disease in certain individuals. Case reports do not prove causal relationships, however, because they do not

provide information concerning the number of people exposed, the incidence rate among the exposed and control groups, or other quantitative information concerning risk.

Retrospective epidemiological studies examine populations who have already acquired the disease of interest. They look back in time to evaluate exposures to suspected risk factors. Retrospective studies may use either a case-control design or a cohort design to provide a comparison group. The major difficulty with retrospective studies is that the injurious exposures occurred in the past, before the scientists started their work. In the case of asbestos-related diseases, the injurious exposures occurred many decades earlier. As a result, the scientists have incomplete information concerning the intensity and duration of exposure. In addition, retrospective studies are often flawed by bias. The types of bias that can impact a retrospective study include confounding bias, observation bias, recall bias and selection bias.

Prospective studies evaluate populations who do not have the disease of interest. Information is gathered about the population (e.g. age distribution, gender, smoking history, etc.), and the population is then followed to determine morbidity and mortality outcomes. Since the scientists are able to observe the population as it ages, they are more able to detect bias problems and to correct for them in the analysis of risk. Prospective studies are the most robust sources of data, but they are time-consuming and expensive. Where rare diseases are concerned (e.g. mesothelioma), prospective studies are often not practical because huge populations would need to be followed for decades in order to have sufficient statistical power to detect risk.

In conducting and evaluating epidemiological studies, epidemiologists must analyze the data according to established methods. These are often referred to as the Bradford-Hill principles of causation. (Hill AB. The environment and disease: association or causation. Proc R Soc Med 1965;58:295-300). The degree of risk is very important in concluding that an exposure

causes the disease. This is often expressed as the relative risk. For example, smoking one pack of cigarettes/day causes a relative risk of lung cancer of 10-fold compared to nonsmokers. This is a very large risk, and unlikely to be affected by bias. Thus, epidemiologists are very confident that smoking causes lung cancer. If the relative risk is 1, or the range of relative risk includes one, there is no statistically significant association between the disease and the risk factor. For example, if the relative risk for a particular risk factor is expressed as 0.8 – 1.2, then there is no statistically significant association between that risk factor and the disease being studied.

In analyzing epidemiological studies it is important to look at the consistency of the results. Scientists do not conclude that a causal relationship exists until there are sufficient data to demonstrate a reasonably stable relative risk. This requires repeated studies, and that takes time.

As an epidemiologist, I am familiar with the epidemiological literature regarding the association between asbestos exposure and the development of disease. Through my experience and training I am able to critically analyze that literature and identify studies which are well founded. I am also able to identify those studies which lack epidemiological significance or validity because they have gaps in either the study design, methods, results or stated conclusions.

VII. Asbestos in the Ambient Air

Asbestos exists in the ambient air throughout the United States. Asbestos fibers have even been detected in the air in remote islands in the Pacific, and the fibers are believed to have been carried there by wind currents. Asbestos has been found in areas in which asbestos has never been commercially used, perhaps due to naturally occurring local outcroppings of asbestos.

People in the general population breathe about 10 million cubic centimeters of air each day. A typical concentration of asbestos in urban air in the United States is 0.0002 f/cc, although

this level of asbestos concentration in the ambient air varies, particularly as between urban and rural areas. Therefore, a person in the general population, with no occupational exposure to asbestos, breathes approximately 2000 asbestos fibers per day. As a result of ambient exposure, all adults in the United States have millions of asbestos fibers in their lungs.

This background level of asbestos exposure causes no ill effect in these individuals. These individuals will suffer no changes on x-ray, pulmonary function tests, or pathology from their exposure to asbestos in the ambient air.

VIII. In Place Asbestos-Containing Building Materials Do Not Pose a Health Risk

There is no valid epidemiological evidence linking exposure to asbestos-containing materials in buildings to asbestos-related disease under normal conditions of use or occupancy. I have not observed any cases of an asbestos-related disease caused by such exposures.

I have never diagnosed or heard of a case of asbestosis developing from even prolonged exposure to asbestos at concentrations of less than several fibers/cc of air. All people living in urban areas have millions of asbestos fibers in their lungs simply by virtue of living in that environment. There is no evidence, however, that consistently breathing such ubiquitous environmental levels of asbestos over an entire lifetime causes an increased incidence of any asbestos-related disease.

Undisturbed in place asbestos-containing building materials do not pose a health risk. This is so because there is no hazard without exposure, and exposure only occurs with inhalation. It is only when respirable asbestos fibers are somehow released from asbestos-containing building materials and inhaled in sufficient quantities over a sufficient period of time that a potential health risk might arise.

Not every exposure to asbestos creates a risk of disease. Whether a person is at increased risk from exposure to asbestos, including exposures from asbestos-containing materials in buildings, depends on the level of exposure or dose, the type of asbestos fibers, and the size of the fibers. There are levels of asbestos exposure below which disease has not been shown to occur. This is true for asbestosis, lung cancer, and mesothelioma.

A. There is No Credible Epidemiological Evidence of Risk from Asbestos-Containing Building Materials

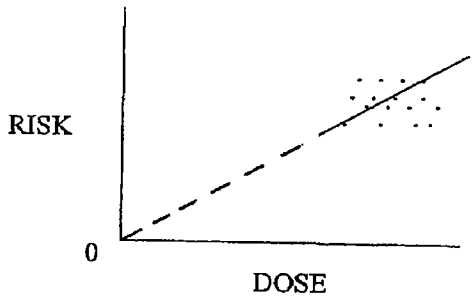
Though there are published studies that have been claimed by some to show an association between asbestos-related disease and exposure to asbestos containing products in buildings, there is no valid epidemiological evidence of such an association. I have examined a number of such studies and I do not find their results to be conclusive that exposure to asbestos from in place asbestos-containing building material in buildings is associated with the development of asbestos-related diseases.

Some of the studies that are the basis for such claims have focused on custodians and janitors. In my opinion those studies are flawed. A major concern I have with these studies is that they have confounding factors, for example, the studies include individuals with asbestos exposures from sources other than the sources being studied. Because of the presence of such confounding factors, the individuals studied were not proper study subjects. Although such studies attempt to show an association between exposure to asbestos containing products in buildings and the incidence of asbestos-related disease in janitors and custodians working in those buildings, many of those janitors and custodians were otherwise occupationally exposed to asbestos at substantial levels before they became janitors or custodians.

B. The Linear No-Threshold Model

Agencies of the U.S. Government, including the USEPA and OSHA, have employed what is called the "linear no-threshold model" to describe the risk associated with asbestos exposure for regulatory purposes. The linear no-threshold model assumes that the risk of developing an asbestos-related disease increases in a linear manner in proportion to the dose and, by definition, presupposes that any exposure to asbestos creates a risk of asbestos-related disease.

The linear no-threshold model is purely theoretical and based on the assumption that the response is linear at all dose levels. The model's essential hypothesis is that any exposure, no matter how small, increases the risk of disease in a linear fashion. The model is illustrated as follows:



The risk axis represents increasing risk, and the dose axis represents increasing dose. The dots represent epidemiological studies which have identified asbestos-disease risks at certain levels of exposure to asbestos. In other words, these dots represent death rates where real people actually developed disease, and died from it. The underlying epidemiological studies that are reflected by the dots, however, demonstrate that risk has only been associated with high levels of exposure. There are no epidemiological studies indicating risk at lower levels of exposure

(hence the absence of dots with low exposures). Despite the absence of disease with low exposures, the linear no-threshold model continues the straight line down to the point where exposure = 0, and risk = 0. This is represented by the broken line. Working backwards from that model, any exposure, no matter how trivial or negligible, could be calculated to cause some degree of risk.

In short, the linear no-threshold model's assumption that a straight line can be drawn from the studies that show risk at high levels of exposure to zero exposure has no scientific support. It is unknown whether the line (below the high level exposure studies) should be curved, or whether it should simply drop off to zero. It is possible that, below a certain level of exposure, the risk is zero. It is also possible that the line should be scooped, resembling a hockey stick, reflecting a zero risk at low dose exposures. At exposure levels that are very low, events become so scarce that they cannot be detected and, it is not feasible to define a specific level at which a risk of asbestos-related disease begins.

Moreover, there is substantial evidence in the medical literature that the linear no-threshold model is not correct. For example, there is considerable evidence that low level exposures to asbestos are tolerated in human beings without any resultant adverse health effects. Residents in the Quebec asbestos mining areas were exposed to elevated levels of chrysotile asbestos (much of which was contaminated with tremolite) for most of the last century. Despite their lifetime exposures to elevated ambient levels of asbestos, and decades of study by researchers interested in the health effects of asbestos exposure, there is no evidence of an increased risk of asbestos-related diseases in those residents. (Churg A., Lung asbestos content in long-term residents of a chrysotile mining town. Am Rev Respir Dis 1986; 134:125-127; Camus M. Siemiatycki J, Meek B., Nonoccupational exposure to chrysotile asbestos and the risk

of lung cancer. N Engl J Med 1998; 338:1565-1571). The EPA's use of the linear no-threshold model to predict mesothelioma in Quebec overestimated the risk by approximately 60-fold. (Camus M, Siemiatycki J, Case BW, et al. Risk of mesothelioma among women living near chrysotile mines versus US EPA asbestos risk model: preliminary findings. Ann Occup Hyg 2002;46:95-98).

The invalidity of the linear no-threshold model is also demonstrated by the consistently low mesothelioma rates in the female population in the United States. In the middle of the twentieth century there was a dramatic change in how women were employed in U.S. society. Women left their homes and began working in cities in ever increasing numbers. Many of those women became office workers in significantly greater number than they had prior to World War II. Thus, for several decades, women have been working in office buildings, where presumably they are exposed to asbestos-containing building materials. Yet the U.S. rate among women of mesothelioma, the sentinel tumor for asbestos, has remained unchanged. There are approximately one to two cases of mesothelioma per million women per year. That rate has been virtually constant for more than 40 years, despite the substantial increase in the number of women employed in jobs outside the home, especially in office buildings.

In contrast, the mesothelioma rate in men changed significantly during that same time period. Men who were in their 20s, or perhaps late teens and 20s during World War II, were heavily exposed to asbestos because of the boom in ship building and commercial construction and in post-war jobs, including as asbestos insulators. In fact, asbestos was heavily used in U.S. industry for years after World War II, declining only in the 1970s. The mesothelioma rate among these men has risen to as high as 20 cases per million per year, a ten-fold rate increase as compared to women.

IX. Threshold Levels for Asbestos-Related Disease

Asbestosis is associated with very high cumulative levels of asbestos exposure that historically occurred almost exclusively in occupational environments. The incidence of asbestosis has declined remarkably in recent years as a result of exposure level restrictions in the workplace that began in earnest around 1970. The amount of asbestos exposure sufficient to cause asbestosis is approximately 25 f/cc-years or more. Even at an exposure of 40 to 100 f/cc years, the life-time risk of asbestosis is only one percent. Most researchers and physicians agree that an individual will not develop asbestosis from a dose below 25 f/cc-years. If an individual worked at a time-weighted average of 0.1 f/cc, it would take that individual 250 years to reach a cumulative dose of 25 f/cc-years.

Asbestos-related lung cancer is almost invariably associated with significant past occupational exposure to asbestos, and most researchers and physicians opine that, in order for an individual to develop an asbestos-related lung cancer, the individual must first have clinical asbestosis. Therefore, an exposure of at least 25 f/cc-years is necessary to put an individual at risk for asbestos-related lung cancer. Asbestos-related lung cancer rarely, if ever, occurs absent a substantial smoking history.

Comparatively speaking, mesothelioma is a lower dose disease than asbestosis and lung cancer. The threshold exposure necessary for amosite and tremolite to cause mesothelioma is 5 f/cc-years. An exposure to asbestos at 0.1 f/cc for approximately 50 years (i.e. 5 f/cc-years) would be necessary to create a risk of mesothelioma from exposure to tremolite. Such a lengthy exposure period would be extremely rare.

Chrysotile asbestos rarely causes mesothelioma, although mesothelioma may occur as a result of the tremolite that may contaminate chrysotile. However, an individual requires

approximately 100 f/cc years exposure to chrysotile contaminated with tremolite in order to be at risk for mesothelioma.

X. Prior Reports

I have previously provided two reports in this matter. The original report is dated October 12, 2005 ("Attachment C"), and the supplemental report is dated January 18, 2006 ("Attachment D"). I hereby incorporate the opinions expressed in those reports.

XI. Reservations

I reserve the right to supplement these opinions as appropriate to address views expressed by any other expert who submits a report or testifies in this case.

I also reserve the right to explicitly address the articles cited by Dr. Laura Welch, and Dr. Henry Anderson, and any other expert who submits a report or testifies in this case.

For the time spent on this matter as a designated expert, I charge my customary rate of $500 per hour. I am also reimbursed for my reasonable and necessary expenses.

William G. Hughson, M.D., D. Phil.